```
### Status: Path 1 of [Dialog Information Services via Modem]

### Status: Initializing TCP/IP using (UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ******** HHHHHHHH SSSSSSSS?
### Status: Signing onto Dialog
 ********
ENTER PASSWORD:
 ******** HHHHHHHH SSSSSSSS? ********
Welcome to DIALOG
### Status: Connected


Dialog level 02.13.02D

Last logoff:  06may03 13:54:37
Logon file001 06may03 15:59:03
KWIC is set to 50.
HILIGHT set on as '*'
* * * * See HELP NEWS 225 for information on new search prefixes
and display codes

***                                    ***


File   1:ERIC  1966-2003/Apr 23
       (c) format only 2003 The Dialog Corporation

      Set  Items  Description
      ---  -----  -----------
Cost is in DialUnits
?b 155, 159, 5, 73
       06may03 15:59:21 User259876 Session D493.1
            $0.29    0.083 DialUnits File1
     $0.29  Estimated cost File1
     $0.06  TELNET
     $0.35  Estimated cost this search
     $0.35  Estimated total session cost   0.083 DialUnits


SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2003/May W1
        (c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession numbers have
changed.  Please see HELP NEWS 155.
  File 159:Cancerlit  1975-2002/Oct
        (c) format only 2002 Dialog Corporation
*File 159: Cancerlit ceases updating with immediate effect.
Please see HELP NEWS.
  File   5:Biosis Previews(R)  1969-2003/Apr W4
        (c) 2003 BIOSIS
*File   5: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  73:EMBASE  1974-2003/Apr W4
        (c) 2003 Elsevier Science B.V.
*File  73: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.

      Set  Items  Description
      ---  -----  -----------
?s (HSE) (s) (tyrosinase (w) promoter) (s) (vector or plasmid)
           2121  HSE
```

```
          12760  TYROSI   E
         301325  PROMOTER
         218319  VECTOR
         184682  PLASMID
      S1      4  (HSE) (S) (TYROSINASE (W) PROMOTER) (S) (VECTOR OR
                 PLASMID)
?rd
...completed examining records
      S2      1  RD (unique items)
?t s2/3,k/all

 2/3,K/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

09550518   21331642   PMID: 11438833
```

A transcriptional feedback loop for tissue-specific expression of highly cytotoxic genes which incorporates an immunostimulatory component.

Emiliusen L; Gough M; Bateman A; Ahmed A; Voellmy R; Chester J; Diaz R M; Harrington K; Vile R

Molecular Medicine Program, Guggenheim 18, Mayo Clinic, Rochester, MN 55905, USA.

Gene therapy (England) Jul 2001, 8 (13) p987-98, ISSN 0969-7128
Journal Code: 9421525
Contract/Grant No.: RO1 CA85931; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

...Therefore, we devised a transcriptional feedback loop to restrict gene expression of very potent genes to melanoma cells. We screened different elements of the human *tyrosinase* *promoter* to find one which gave no detectable expression in non-melanoma cells but was active in melanoma cell lines. This weak, but highly tissue specific, element (Tyr-300) was then used as the basis for a transcriptional amplification feedback loop in which a consensus heat shock element (*HSE*) was cloned upstream of Tyr-300. The cytotoxic gene was cloned downstream of the *HSE*-Tyr-300 element along with a mutated form of the heat shock factor-1 (HSF-1) transcription factor, which no longer requires cellular stress to...

... expression of both the cytotoxic and the HSF-1 genes in melanoma cells. Gradual build up of HSF-1 amplified expression through binding to the *HSE* to give levels of cytotoxicity similar to that provided by a CMV promoter. However, no leakiness was observed in multiple non-melanoma cell lines tested...

... is a highly immunostimulatory event which enhances the antitumour vaccination effects of direct tumour cell destruction. Having demonstrated the compatibility of the component elements in *plasmid* form, we incorporated the feedback loop into a hybrid LTR-modified retroviral *vector* and confirmed that the system can be effective in the form of a viral *vector* . The format of the feedback loop described here could be exploited for any tissue type in which a highly tissue-specific element can be identified...

```
?ds

Set     Items   Description
S1          4   (HSE) (S) (TYROSINASE (W) PROMOTER) (S) (VECTOR OR PLASMID)
S2          1   RD (unique items)
?s (HSE) (s) (promoter) (s) (vector or plasmid)
          2121  HSE
         301325  PROMOTER
         218319  VECTOR
         184682  PLASMID
      S3     58  (HSE) (S) (PROMOTER) (S) (VECTOR OR PLASMID)
```

```
?s s3 and (HSF)
              58  S3
            2520  HSF
      S4      17  S3 AND (HSF)
?rd s4
...completed examining records
      S5       5  RD S4 (unique items)
?t s5/3,k/all
```

5/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

14378239 22348180 PMID: 12462526

Gene expression of 70 kDa heat shock protein of Candida albicans: transcriptional activation and response to heat shock.

Sandini S; Melchionna R; Bromuro C; La Valle R; et al
Laboratory of Bacteriology and Medical Mycology, Istituto Superiore di Sanita, Viale Regina Elena, 299, 00161 Rome, Italy.

Medical mycology - official publication of the International Society for Human and Animal Mycology (England) Oct 2002, 40 (5) p471-8, ISSN 1369-3786 Journal Code: 9815835
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... was cloned and sequenced. It contains at least three heat shock elements (HSEs), specific DNA sequences that are bound by the heat shock transcription factor (*HSF*), and one stress response element (STRE), which is an upstream activator sequence (UAS) that causes transcription activation under stress. The binding of *HSF* to *HSE* in the CaHSP70 *promoter* region is constitutive, although the mobility of protein/DNA complexes is altered after heat shock. The CaHSP70 *promoter* was cloned into a lacZ reporter *plasmid*, and was able to respond to heat shock in C. albicans as well as in Saccharomyces cerevisiae.

5/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

10710636 97059999 PMID: 8904320

Stable overexpression of human *HSF* -1 in murine cells suggests activation rather than expression of *HSF*-1 to be the key regulatory step in the heat shock gene expression.

Mivechi N F; Shi X Y; Hahn G M
Cancer Biology Research Laboratory, Department of Radiation Oncology, Stanford University School of Medicine, CA 94305, USA.

Journal of cellular biochemistry (UNITED STATES) Oct 1995, 59 (2) p266-80, ISSN 0730-2312 Journal Code: 8205768
Contract/Grant No.: CA 54093; CA; NCI; PO1 CA-44665; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Stable overexpression of human *HSF* -1 in murine cells suggests activation rather than expression of *HSF*-1 to be the key regulatory step in the heat shock gene expression.

Transcription of the heat shock genes is regulated by the activation of the heat shock transcription factor (*HSF*-1). After heat shock, *HSF*-1 forms oligomers and binds to the heat shock element (*HSE*), which consists of several repeats of NGAAN located in the *promoter* region of the heat shock genes. *HSF* -1 is then phosphorylated, leading to the enhanced transcription of the heat shock genes likely by transactivation. We have

stably overexpressed th[redacted]uman heat shock transcription tor-1 (*HSF*-1) in murine cells to investigate whether the regulation of the expression of the heat shock genes may partly reside at the level of *HSF*-1 expression. Human *HSF*-1 cDNA was cloned into a retroviral *vector* (pvhhsf-1) and was overexpressed in a murine fibroblast cell line. The overexpressed human *HSF* -1 is found in both the cytoplasm and nucleus of control cells but is translocated into the nucleus upon heat shock. Electrophoretic mobility shift analysis suggests that the human *HSF*-1 has constitutive DNA binding ability and its DNA binding ability is increased upon heat shock. Cross-linking experiments indicate that the overexpressed human *HSF*-1 is mainly a monomer under control conditions and forms oligomers upon heat shock. Immunoblotting shows that the human *HSF*-1 is phosphorylated upon heat shock and its apparent molecular weight is shifted up by at least 10 kDa. In spite of both the DNA binding ability and phosphorylation, the overexpression of human *HSF* -1 does not increase the transcription of murine HSP-70 mRNA or increase the synthesis of other HSPs after heat shock beyond that observed in...

... and an apparent lack of induction of one HSP-70 kDa species when the protein pattern is analyzed by isoelectric focusing. Interestingly, cells overexpressing human *HSF*-1 show a 4-fold increase in the basal expression of luciferase when the plasmids containing the human HSP-70 *promoter* ligated to the luciferase reporter gene are transiently expressed in these cells. Murine cells overexpressing human *HSF*-1 are more resistant to the cytotoxic effects of heat when compared to the control untransfected cells, but the kinetics of thermotolerance development and decay is similar between *HSF*-1 transfected and untransfected cells. In conclusion, human *HSF* -1 protein in murine fibroblasts is modified in a similar fashion as the endogenous mouse *HSF*-1 after heat shock. However, the overexpression of *HSF* -1 does not result in overproduction of heat shock proteins after heat shock, perhaps because these cells contain abundant amounts of endogenous *HSF*-1.

5/3,K/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

09550518 21331642 PMID: 11438833

A transcriptional feedback loop for tissue-specific expression of highly cytotoxic genes which incorporates an immunostimulatory component.

Emiliusen L; Gough M; Bateman A; Ahmed A; Voellmy R; Chester J; Diaz R M; Harrington K; Vile R

Molecular Medicine Program, Guggenheim 18, Mayo Clinic, Rochester, MN 55905, USA.

Gene therapy (England) Jul 2001, 8 (13) p987-98, ISSN 0969-7128 Journal Code: 9421525

Contract/Grant No.: RO1 CA85931; CA; NCI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

...we devised a transcriptional feedback loop to restrict gene expression of very potent genes to melanoma cells. We screened different elements of the human tyrosinase *promoter* to find one which gave no detectable expression in non-melanoma cells but was active in melanoma cell lines. This weak, but highly tissue specific, element (Tyr-300) was then used as the basis for a transcriptional amplification feedback loop in which a consensus heat shock element (*HSE*) was cloned upstream of Tyr-300. The cytotoxic gene was cloned downstream of the *HSE*-Tyr-300 element along with a mutated form of the heat shock factor-1 (*HSF*-1) transcription factor, which no longer requires cellular stress to activate its trimerisation, nuclear localisation and transcriptional activation properties. Low levels of expression from Tyr-300 initiated expression of both the cytotoxic and the *HSF*-1 genes in melanoma cells. Gradual build

up of *HSF*-1 amplifi expression through binding to e *HSE* to give levels of cytotoxicity similar to that provided by a CMV *promoter*. However, no leakiness was observed in multiple non-melanoma cell lines tested. In addition to amplifying low levels of weak tissue-specific expression, the use of *HSF* -1 also leads to activation of endogenous stress-related genes such as hsp70. Induction of these genes, in the presence of cell killing by the...

... is a highly immunostimulatory event which enhances the antitumour vaccination effects of direct tumour cell destruction. Having demonstrated the compatibility of the component elements in *plasmid* form, we incorporated the feedback loop into a hybrid LTR-modified retroviral *vector* and confirmed that the system can be effective in the form of a viral *vector* . The format of the feedback loop described here could be exploited for any tissue type in which a highly tissue-specific element can be identified...

5/3,K/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

08022791 94088515 PMID: 8264586
Heat shock factor can activate transcription while bound to nucleosomal DNA in Saccharomyces cerevisiae.
Pederson D S; Fidrych T
Department of Microbiology and Molecular Genetics, University of Vermont School of Medicine, Burlington 05405.
Molecular and cellular biology (UNITED STATES) Jan 1994, 14 (1) p189-99, ISSN 0270-7306 Journal Code: 8109087
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

After each round of replication, new transcription initiation complexes must assemble on *promoter* DNA. This process may compete with packaging of the same *promoter* sequences into nucleosomes. To elucidate interactions between regulatory transcription factors and nucleosomes on newly replicated DNA, we asked whether heat shock factor (*HSF*) could be made to bind to nucleosomal DNA in vivo. A heat shock element (*HSE*) was embedded at either of two different sites within a DNA segment that directs the formation of a stable, positioned nucleosome. The resulting DNA segments were coupled to a reporter gene and transfected into the yeast Saccharomyces cerevisiae. Transcription from these two *plasmid* constructions after induction by heat shock was similar in amount to that from a control *plasmid* in which *HSF* binds to nucleosome-free DNA. High-resolution genomic footprint mapping of DNase I and micrococcal nuclease cleavage sites indicated that the *HSE* in these two plasmids was, nevertheless, packaged in a nucleosome. The inclusion of *HSE* sequences within (but relatively close to the edge of) the nucleosome did not alter the position of the nucleosome which formed with the parental DNA fragment. Genomic footprint analyses also suggested that the *HSE* -containing nucleosome was unchanged by the induction of transcription. Quantitative comparisons with control plasmids ruled out the possibility that *HSF* was bound only to a small fraction of molecules that might have escaped nucleosome assembly. Analysis of the helical orientation of *HSE* DNA in the nucleosome indicated that *HSF* contacted DNA residues that faced outward from the histone octamer. We discuss the significance of these results with regard to the role of nucleosomes in...

5/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.

07543640 92412106 PM[redacted] 1356336

Activation of human multidrug resistance-1 gene promoter in response to heat shock stress.

Miyazaki M; Kohno K; Uchiumi T; Tanimura H; Matsuo K; Nasu M; Kuwano M
Department of Biochemistry, Oita Medical University, Japan.

Biochemical and biophysical research communications (UNITED STATES) Sep 16 1992, 187 (2) p677-84, ISSN 0006-291X Journal Code: 0372516
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

...expression, we have established human cancer KB cell lines which could stably integrate bacterial chloramphenicol acetyltransferase (CAT) gene driven by various lengths of the MDR1 *promoter*. Kst-6 has an integrated *plasmid*, pMDRCAT1, containing the human MDR1 *promoter* of -2 kilobases. The MDR1 gene *promoter* contains a typical heat shock element (*HSE*) motif located -152 bp to -178 bp from the initiation site. Heat shock at 45 degrees C for 90 min significantly induced CAT activity in Kst-6 cells. Northern blot analysis showed a 4-5 fold increase in CAT mRNA levels in Kst-6 cells. Deletion analysis of the MDR1 *promoter* demonstrated that the induction of CAT activity was observed in Kxh-14 cells containing a *HSE* -deleted MDR1 *promoter* construct, pMDRCAT7. However, further deletion analysis showed that heat shock could not induce CAT activity in Khp-1 cells containing -76 approximately +121 base sequence of the *promoter*, suggesting that a new heat shock responsible element was located at between -136 and -76. Gel shift assay showed that the heat shock factor (*HSF*) could bind to the *HSE* motif located at -152 bp to -178 bp in the MDR1 *promoter* . We also found that one distinct DNA-protein complex formed specifically within the MDR1 *promoter* region -99 to -66 was not significantly increased, but relatively more stabilized under mild denaturing condition in the nuclear extract of heat-shocked cells. In our present assay system, activation of the MDR1 *promoter* in response to heat shock appears to be mediated through both a new heat shock responsive element and MDR1 specific transcription factor.

?ds

Set	Items	Description
S1	4	(HSE) (S) (TYROSINASE (W) PROMOTER) (S) (VECTOR OR PLASMID)
S2	1	RD (unique items)
S3	58	(HSE) (S) (PROMOTER) (S) (VECTOR OR PLASMID)
S4	17	S3 AND (HSF)
S5	5	RD S4 (unique items)

```
?s ((tissue or cell or tumor or tumour) (w) specific (w) promoter)
Processing
         2555559  TISSUE
         6749444  CELL
         2119174  TUMOR
          270811  TUMOUR
         2572849  SPECIFIC
          301325  PROMOTER
      S6    1004  ((TISSUE OR CELL OR TUMOR OR TUMOUR) (W) SPECIFIC (W)
                  PROMOTER)
?s s6 and (amplification (w) promoter (w) element)
            1004  S6
          176997  AMPLIFICATION
          301325  PROMOTER
          215726  ELEMENT
               0  AMPLIFICATION(W)PROMOTER(W)ELEMENT
      S7       0  S6 AND (AMPLIFICATION (W) PROMOTER (W) ELEMENT)
?s s6 (s) (HSE)
            1004  S6
            2121  HSE
      S8       0  S6 (S) (HSE)
?s s6 and (HSE)
            1004  S6
```

```
            2121  HSE
      S9       0  S6 AND (HSE)
?ds

Set     Items   Description
S1          4   (HSE) (S) (TYROSINASE (W) PROMOTER) (S) (VECTOR OR PLASMID)
S2          1   RD (unique items)
S3         58   (HSE) (S) (PROMOTER) (S) (VECTOR OR PLASMID)
S4         17   S3 AND (HSF)
S5          5   RD S4 (unique items)
S6       1004   ((TISSUE OR CELL OR TUMOR OR TUMOUR) (W) SPECIFIC (W) PROM-
               OTER)
S7          0   S6 AND (AMPLIFICATION (W) PROMOTER (W) ELEMENT)
S8          0   S6 (S) (HSE)
S9          0   S6 AND (HSE)
?logoff
       06may03 16:11:34 User259876 Session D493.2
            $2.71    0.848 DialUnits File155
               $1.26  6 Type(s) in Format  3
            $1.26  6 Types
     $3.97  Estimated cost File155
            $0.83    0.281 DialUnits File159
     $0.83  Estimated cost File159
            $4.03    0.719 DialUnits File5
     $4.03  Estimated cost File5
            $8.34    0.902 DialUnits File73
     $8.34  Estimated cost File73
            OneSearch, 4 files,  2.750 DialUnits FileOS
     $3.02  TELNET
    $20.19  Estimated cost this search
    $20.54  Estimated total session cost   2.833 DialUnits


### Status: Signed Off. (13 minutes)
```